```
          PLURAL? OR MULTI?)(3A)(DYE OR LABEL? OR TAG OR INDICATOR))
          E BLUMENTHAL D/AU
L4   105 S E3,E8,E22-26
L5     0 S L4 AND(BILAB? OR DILAB? OR MULTILAB? OR (DUAL OR BI OT TWO OR 2 OR
          PLURAL? OR MULTI?)(3A)(DYE OR LABEL? OR TAG OR INDICATOR))
L6     2 S L4 AND(DONOR OR ACCEPT?)
L7     0 S (NONFLUORES? OR NON FLUORES?)(4A)(COMPLEX? OR PAIR? OR ASSOCIAT? OR
          CONTACT?) AND L4
L8     2 S L4 AND QUENCH?
L9    22 S L2-3
L10   12 S L9 AND PY<2000
L11    4 S L6,L8
      FILE 'BIOSIS' ENTERED AT 12:41:04 ON 20 DEC 2006
L12    3 S L10-11
      FILE 'MEDLINE' ENTERED AT 12:41:30 ON 20 DEC 2006
L13    9 S L10-11
      FILE 'CA, BIOSIS, MEDLINE' ENTERED AT 12:42:37 ON 20 DEC 2006
L14   22 DUP REM L10 L11 L12 L13 (6 DUPLICATES REMOVED)

=> log y
STN INTERNATIONAL LOGOFF AT 12:43:18 ON 20 DEC 2006

=> d his

      (FILE 'HOME' ENTERED AT 13:23:01 ON 20 DEC 2006)
      FILE 'CA' ENTERED AT 13:23:18 ON 20 DEC 2006
L1 400160 S (KINASE OR PHOSPHATASE OR PHOSPHORYLASE)
L2    1039 S HGC OR CHORIONIC GONADOTROPHIN
L3      40 S L1 AND L2
L4       2 S (HGC OR CHORIONIC GONADOTROPHIN)(3A)(PHOSPHORYL? OR PHOSPHAT?)
L5   15734 S L1(4A)(PEPTIDE OR SUBSTRATE)
L6     788 S L5 AND FLUORESC?
L7    1276 S (DYE OR LABEL? OR TAG OR INDICATOR)(5A)(DIMER OR STACK?)
L8       2 S L6 AND L7
L9     727 S (BILAB? OR DILAB? OR MULTILAB? OR (DUAL OR 2 OR PLURAL? OR MULTI?
          OR DOUBLE)(3A)(DYE OR LABEL? OR TAG OR INDICATOR))(4A)(PEPTIDE OR
          SUBSTRATE)
L10      60 S L1 AND L9
L11       5 S L7 AND L9
L12     106 S L3-4,L8,L10-11
L13      66 S L12 AND PY<2000
      FILE 'BIOSIS' ENTERED AT 13:45:59 ON 20 DEC 2006
L14      68 S L13
      FILE 'MEDLINE' ENTERED AT 13:46:53 ON 20 DEC 2006
L15      80 S L13
      FILE 'CA, BIOSIS, MEDLINE' ENTERED AT 13:47:38 ON 20 DEC 2006
L16     136 DUP REM L13 L14 L15 (78 DUPLICATES REMOVED)

=> d bib,ab,kwic l16 1-136

L16  ANSWER 6 OF 136  CA  COPYRIGHT 2006 ACS on STN
AN   130:11982  CA
TI   Fluorogenic protease substrates based on dye-dimerization
IN   Wei, Ai-Ping; Williams, Michael G.
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PA Minnesota Mining and Manufacturing Co., USA
SO PCT Int. Appl., 27 pp.
PI WO 9850579 A1 19981112 WO 1997-US16579 19970908
PRAI US 1997-846828 A 19970501
AB A method of biol. assay comprises the steps of providing an enzyme **substrate** comprising **2** fluorescence **dye** groups bound to a **peptide**, the dye groups being of proximity sufficiently close so as to essentially self-quench fluorescence of the dye groups, wherein self-quenching of fluorescence of the **dye** groups is effected by **dye stacking**, and enzymically cleaving the peptide to release the fluorescence **dye** groups from **dye stacking**, and producing an increase in fluorescence intensity. A protease substrate I (TMR-Val-Pro-Arg-Gly-Lys-TMR, TMR = tetramethylrhodamine) for use in the method of the invention is also disclosed. For a wide spectrum of excitation frequencies, fluorescence intensity of the cleaved substrate soln. is as much as 29-fold that of the intact substrate soln., averaging from 25-28-fold the intensity, for emission wavelengths from 570 to 585 nm, a range easily visible to the human eye. This invention finds use in detection and identification of microorganisms, sterilization assurance, pharmaceutical discovery, enzyme assays, immunoassays, and other biol. assays. Use of the fluorogenic protease substrate I is demonstrated for the detection of Vibrio parahaemolyticus.

L16 ANSWER 36 OF 136 CA COPYRIGHT 2006 ACS on STN
AN 120:265157 CA
TI Antibody-mediated fluorescence enhancement based on shifting the intramolecular **dimer** ⇔ monomer equilibrium of fluorescent **dyes**
AU Wei, Ai-Ping; Blumenthal, Donald K.; Herron, James N.
CS Departments of Pharmaceutics, University of Utah, Salt Lake City, UT, 84108, USA
SO Analytical Chemistry (**1994**), 66(9), 1500-6
AB A novel concept is described for directly coupling fluorescence emission to protein-ligand binding. It is based on shifting the intramol. monomer ⇔ **dimer** equil. of two fluorescent **dyes** linked by a short spacer. A 13-residue peptide, recognized by a monoclonal antibody against human chorionic gonadotropin (hCG), was labeled with fluorescein (F) and tetramethylrhodamine (T) at its N- and C-termini, resp. Spectral evidence suggests that when the conjugate is free in soln., F and T exist as an intramol. dimer. Fluorescence quenching of fluorescein and rhodamine is □98% and □90%, resp., due to dimerization. When the **double-labeled peptide** is bound to anti-hCG, however, the rhodamine fluorescence increases by ≤7.8-fold, depending upon the excitation wavelength. This is attributed to the dissocn. of intramol. dimers brought about by conformational changes of the conjugate upon binding. Fluorescein fluorescence was still quenched because of excited-state energy transfer and residual ground-state interactions. Antibody binding also resulted in a □3.4-fold increase in fluorescence anisotropy of the peptide. These changes in intensity and anisotropy allow direct measurement of antigen-antibody binding with a fluorescence plate reader or a polarization analyzer, without the need for sepn. steps and labeling antibodies. Because recent advances in peptide technol. have allowed rapid and economical identification of antigen-mimicking **peptides**, the **double-labeled peptide** approach offers many opportunities

for developing new diagnostic assays and screening new therapeutic drugs. It also has many potential applications to techniques involving recombinant antibodies, biosensors, cell sorting, and DNA probes.

L16 ANSWER 58 OF 136 CA COPYRIGHT 2006 ACS on STN
AN 115:109008 CA
TI A continuous fluorescence assay for protein **kinase** C
AU McIlroy, Brian K.; Walters, John D.; Johnson, J. David
CS Coll. Dent., Ohio State Univ., Columbus, OH, 43210, USA
SO Analytical Biochemistry (**1991**), 195(1), 148-52
AB A 6-acryloyl-**2**-dimethylaminoapthalene (acrylodan)-**labeled** 25-amino acid **peptide** (acrylodan-CKKKKRFSFKKSFKLSGFSFKKNKK-COO-), contg. the protein **kinase** C (PKC) phosphorylation sites of brain myristoylated alanine-rich **kinase** C substrate (MARCK) protein, undergoes a 20% fluorescence decrease when it is phosphorylated by PKC. This fluorescence decrease is dependent on the presence of PKC, Ca2+ (half-maximal stimulation at pCa = 6.2), phosphatidylserine, diacylglycerol, or phorbol-12-myristate-13-acetate (half-maximal stimulation at 2 nM) and ATP, and correlates well (r = 0.007) with [32P]phosphate incorporation into the peptide. This fluorescence assay allows detection of 0.02 nM PKC, whereas similar concns. of cAMP-dependent or type II calmodulin-dependent protein **kinases** produced no change in peptide fluorescence. The method can be used to assay purified PKC as well as activity in crude brain homogenates. Incubation of PKC with staurosporine inhibits the fluorescence decrease with an IC50 of 2 nM. Thus, the fluorescence decrease that occurs in the acrylodan-peptide provides a continuous fluorescence assay for PKC activity.

L16 ANSWER 66 OF 136 CA COPYRIGHT 2006 ACS on STN
AN 113:188695 CA
TI Phorbol ester-stimulated phosphorylation of keratinocyte transglutaminase in the membrane anchorage region
AU Chakravarty, Rupa; Rong, Xianhui; Rice, Robert H.
CS Charles A. Dana Lab. Toxicol., Harvard Sch. Public Health, Boston, MA, 02115, USA
SO Biochemical Journal (**1990**), 271(1), 25-30
AB The membrane-bound transglutaminase of cultured keratinocytes became radioactively labeled on addn. of 32P-labeled inorg. phosphate to the medium. Transglutaminase phosphorylation was also demonstrable using particulate material isolated from cell homogenates. Compatible with mediation of the labeling by protein **kinase** C, the degree of phosphorylation in intact cells was simulated ≈5-fold in 4 h on treatment with the tumor-promoting phorbol ester PMA, but not by phorbol. The extent of labeling was virtually unaffected by cycloheximide inhibition of protein synthesis, indicating that it arose primarily through turnover of phosphate in the membrane-bound enzyme. Phosphoamino acid anal. detected labeling only of serine residues. Most of the label was removed by trypsin release of the enzyme from the particulate fraction of cell homogenates, which deletes a membrane anchorage region of ≈10 kDa. On trypsin treatment of the enzyme after immunopptn., the phosphate label was recovered in sol. peptide material with a size of several thousand Da or less. Indicative of fragmentation of the membrane anchorage region, this material was separable by HPLC

into 2 equally **labeled peptides**. Moreover, when the enzyme was labeled with [3H]palmitate or [3H]myristate, the fatty acid-labeled peptide material required nonionic detergent for solubilization and was separable from the phosphate-labeled material by gel filtration. Phorbol ester treatment of cultured keratinocytes in high- or low-Ca2+-contg. medium was not accompanied by an appreciable protein synthesis-independent change in transglutaminase activity. Independent of possible alteration of the intrinsic catalytic activity of the enzyme, phosphorylation may well modulate its interaction with substrate proteins, a potential site for physiol. regulation.

L16 ANSWER 69 OF 136 CA COPYRIGHT 2006 ACS on STN
AN 110:54860 CA
TI In vivo phosphorylation of distinct domains of the 70-kilodalton neurofilament subunit involves different protein **kinases**
AU Sihag, Ram K.; Nixon, Ralph A.
CS Mailman Res. Cent., McLean Hosp., Belmont, MA, 02178, USA
SO Journal of Biological Chemistry (**1989**), 264(1), 457-64
AB A combination of in vivo and in vitro approaches were used to characterize phosphorylation sites on the 70,000-kilodalton (kDa) subunit of neurofilaments (NF-L) and to identify the protein **kinases** that are likely to mediate these modifications in vivo. Neurofilament proteins in a single class of neurons, the retinal ganglion cells, were pulse-labeled in vivo by injecting mice intravitreously with [32P] orthophosphate. Radiolabeled neurofilaments were isolated after they had advanced along optic axons, and the individual subunits were sepd. on SDS-polyacrylamide gels. Two-dimensional chymotryptic phosphopeptide map anal. of NF-L revealed 3 phosphorylation sites: an intensely labeled **peptide** (L-1) and **2** less intensely **labeled peptides** (L-**2** and L-3). Chymotryptic peptide L-1 was identified as the 11-kDa segment contg. the C-terminus of NF-L. The ability of these peptides to serve as substrates for specific protein **kinases** were examd. by incubating neurofilament prepns. with [γ-32P]ATP in the presence of purified cAMP-dependent protein **kinase** or appropriate activators and/or inhibitors of endogenous cytoskeleton-assocd. protein **kinases**. The heparin-sensitive, Ca2+ and cyclic nucleotide-independent **kinase** assocd. with the cytoskeleton selectively phosphorylated L-1 and L-3 but had little, if any, activity toward L-2. When this **kinase** was inhibited with heparin, cAMP addn. to the neurofilament prepn. stimulated the phosphorylation of L-2, and addn. of the purified catalytic subunit of cAMP-dependent protein **kinase** induced intense labeling of L-2. At higher labeling efficiencies, the exogenous **kinase** also phosphorylated L-3 and several sites at which labeling was not detected in vitro; however, L-1 was not a substrate. Ca2+ and calmodulin added to neurofilament prepns. in the presence of heparin modestly stimulated the phosphorylation of L-1 and L-3, but not L-2, and the stimulation was reversed by trifluoperazine. The selective phosphorylation of different polypeptide domains on NF-L by 2nd messenger-dependent and -independent **kinases** suggested multiple functions for phosphate groups on this protein.

L16 ANSWER 71 OF 136 CA COPYRIGHT 2006 ACS on STN
AN 110:132824 CA
TI The two major phosphoproteins in growth cones are probably identical to

two protein **kinase** C substrates correlated with persistence of long-term potentiation

AU Nelson, Robert B.; Linden, David J.; Hyman, Carolyn; Pfenninger, Karl H.; Routtenberg, Aryeh

CS Cresap Neurosci. Lab., Northwestern Univ., Evanston, IL, 60201, USA

SO Journal of Neuroscience (**1989**), 9(2), 381-9

AB Because protein **kinase** C (PKC) has been implicated in neurite outgrowth and is present at high levels in growth cone-rich areas of fetal brain, PKC substrates were investigated in a prepn. of isolated nerve growth cone fragments from fetal rat brain and compared with PKC substrates found in adult rat hippocampus. Four major proteins in the growth cone prepn. showed endogenous phosphorylation levels at least 10-fold greater than any other phosphoproteins. Three of these 4 phosphoproteins, termed pp408, pp46, and pp80 (Katz, F. L.; et al., 1985), were phosphorylated by exogenous PKC in a dose-dependent manner, indicating that PKC activity might be of particular importance relative to other **kinases** in growth cone function. The **2** most highly **labeled** PKC **substrates**, pp46 and pp80, comigrated on 2-dimensional gels with the adult hippocampal proteins F1 and 80k (Mr 78-80 kDa, pI 4.0), resp. In addn., similarities in charge heterogeneity, 2-dimensional phosphopeptide maps, and increased phosphorylation in the presence of exogenous PKC or PKC stimulators suggest that protein F1 and 80k are highly homologous to, if not identical to, pp46 and pp80, resp. The relationship of 80k phosphorylation to persistence of long-term potentiation (LTP) has been masked previously by its comigration on 1-dimensional SDS gels with the major phosphoproteins synapsin Ia and Ib. Quant. anal. of in vitro labeled phosphoproteins sepd. by 2-dimensional gel electrophoresis revealed that this 2nd major neural PKC substrate, along with protein F1, was directly correlated with persistence of LTP induced in vivo. Apparently, PKC and its substrates play a central role in growth cone function, and this protein **kinase** system may underlie both normal neurite growth in developing brain and neural plasticity at adult synapses.

L16 ANSWER 119 OF 136 BIOSIS on STN

AN 1979:146136 BIOSIS

TI THE ISOLATION AND PARTIAL CHARACTERIZATION OF LOW MOLECULAR WEIGHT PHOSPHORYLATED COMPONENT OF THE NONHISTONE PROTEINS OF MOUSE NUCLEI.

AU MACGILLIVRAY A J [Reprint author]; JOHNSTON C; MACFARLANE R; RICKWOOD D

CS BIOCHEM LAB, SCH BIOL SCI, UNIV SUSSEX, FALMER, BRIGHTON, SUSSEX BN1 9QG, ENGL, UK

SO Biochemical Journal, (**1978**) Vol. 175, No. 1, pp. 35-46.

AB After labeling of mouse liver nuclei with [γ-32P]ATP in vitro, 10-20% of the radioactivity incorporated into the saline-soluble nuclear and HAP2 chromatin fractions was located in a low MW component (component 10) with an isoelectric point near 4.5 in urea. By using combinations of ion-exchange chromatography, preparative thin-layer isoelectric focusing and gel filtration, this component was isolated from both nuclear fractions. Recovery from the saline-soluble fraction was poor under conditions that allow endogenous **phosphatases** to be active. Component 10 was a phosphoprotein on the basis of enzyme-digestion experiments and the detection of phosphoserine and phosphothreonine. The 32P radioactivity did not appear to be associated with phosphorylated basic

amino acids. Its MW was determined by gel chromatography and electrophoresis in sodium dodecyl sulphate/polyacrylamide gels as approximately 10,000, and tryptic digestion of the reduced carboxymethylated proteins in urea yielded **2** 32P-**labeled peptides**. No function has yet been assigned to component 10, though its similarity to other low MW acidic proteins is discussed.

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=> log y
STN INTERNATIONAL LOGOFF AT 13:49:49 ON 20 DEC 2006

=> d his

     (FILE 'HOME' ENTERED AT 13:58:41 ON 20 DEC 2006)
     FILE 'CA' ENTERED AT 13:58:48 ON 20 DEC 2006
L1 400160 S (KINASE OR PHOSPHATASE OR PHOSPHORYLASE)
L2   1276 S (DYE OR LABEL? OR TAG OR INDICATOR)(5A)(DIMER OR STACK?)
L3    194 S ((BI OR TWO)(3A)(DYE OR LABEL? OR TAG OR INDICATOR))(4A)(PEPTIDE
          OR SUBSTRATE)
L4     22 S L1-2 AND L3
L5     11 S L4 AND PY<2000
     FILE 'BIOSIS' ENTERED AT 14:01:58 ON 20 DEC 2006
L6     20 S L5
     FILE 'MEDLINE' ENTERED AT 14:02:14 ON 20 DEC 2006
L7     25 S L5
     FILE 'CA, BIOSIS, MEDLINE' ENTERED AT 14:02:49 ON 20 DEC 2006
L8     31 DUP REM L5 L6 L7 (25 DUPLICATES REMOVED)

=> d bib,ab l8 1-31

L8   ANSWER 2 OF 31  CA  COPYRIGHT 2006 ACS on STN
AN   127:3284  CA
TI   Identification of in vivo phosphorylation sites of CD45 protein-tyrosine
     phosphatase in 70Z/3.12 cells
AU   Kang, Sanmao; Liao, Pao-Chi; Gage, Douglas A.; Esselman, Walter J.
CS   Department of Microbiology, Michigan State University, East Lansing, MI,
     48824-1101, USA
SO   Journal of Biological Chemistry (1997), 272(17), 11588-11596
AB   Phosphorylation of CD45, a transmembrane protein-tyrosine phosphatase
     (PTPase), has been proposed to mediate docking of signaling proteins and
     to modulate PTPase activity. To study the role of phosphorylation in
     CD45, in vivo phosphorylation sites of CD45 from 70Z/3.12 cells were
     identified using 32P labeling, trypsin digestion, two-dimensional
     peptide mapping, high performance liq. chromatog., phosphoamino acid
     anal., matrix-assisted laser desorption/ionization mass spectrometry,
     and specific enzymic degrdn. Eight phosphopeptides, a through h, were
     isolated and four phosphorylation sites were identified. All four
     phosphorylation sites were in the membrane-distal PTPase domain (D2) and
     the C-terminal tail, and none were in the membrane-proximal PTPase
     domain (D1). One site, Ser(P)939 peptide h, was in the D2 domain and,
     by comparison to the three-dimensional structure of PTP1B, is predicted
     to lie at the apex of the substrate binding loop. Ser939 was the only
     in vitro phosphorylation site for protein kinase C among the
     phosphorylation sites identified. Four of the C-terminal peptides
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identified (d, e, f, and g) spanned the same sequence and were derived from the same phosphorylation site in the C-terminal tail, Ser1204. Peptide a was derived from the intact C terminus and comprised a mixt. of monophosphorylated peptides contg. either Ser(P)1248 or Thr(P)1246. Knowledge of the precise phosphorylation sites of CD45 will lead to the design of expts. to define the role of phosphorylation in PTPase activity and in signaling.

L8 ANSWER 3 OF 31 CA COPYRIGHT 2006 ACS on STN
AN 126:44278 CA
TI A peptide model for calmodulin trapping by calcium/calmodulin-dependent protein **kinase** II
AU Putkey, John A.; Waxham, M. Neal
CS Department Biochemistry Molecular Biology Neurobiology Anatomy, University Texas Medical School, Houston, TX, 77225, USA
SO Journal of Biological Chemistry (**1996**), 271(47), 29619-29623
AB Autophosphorylation of Ca2+/calmodulin-dependent protein **kinase** II (CaM-**kinase**) induces a more than 1000-fold increase in calmodulin (CaM)-binding affinity by dramatically decreasing the off-rate for CaM. In this report, we investigate the mol. mechanism for this phenomenon by comparing the rate of dissocn. of a novel fluorescently **labeled** CaM from **two** synthetic **peptides** and from the phosphorylated and nonphosphorylated forms of a recombinant prepn. of CaM-**kinase**. Dissocn. of a complex of CaM and CKII(296-312), a peptide representing close to the min. CaM-binding domain of the α subunit of CaM-**kinase**, exhibited a fast off-rate of 5.0 s-1. This was similar to the off-rate of 1.1 s-1 for the dissocn. of CaM from the nonphosphorylated form of CaM-**kinase**. In contrast, dissocn. of CaM from either autophosphorylated CaM-**kinase** or peptide CKII(290-314) was extremely slow with apparent off-rates of about 3-9x10-5 s-1. Along with information from the crystal structure of Ca2+/CaM bound to CKII(290-314) (Meador, W. E., Means, A. R., and Quiocho, F. A. (1993) Science 262, 1718-1721), our results suggest a model in which CaM-dependent autophosphorylation of CaM-**kinase** induces a conformational change in the region of the CaM-binding domain which allows the formation of addnl. stabilizing interactions with CaM. We predict that this involves amino acids 293-298 in CaM-**kinase**. The possible consequences of these observations on the reversibility of CaM trapping in native CaM-**kinase** are discussed.

L8 ANSWER 5 OF 31 BIOSIS on STN
AN 1995:407610 BIOSIS
TI Direct identification of a polyamine binding domain on the regulatory subunit of the protein **kinase** casein **kinase** 2 by photoaffinity labeling.
AU Leroy, Didier; Schmid, Nathalie; Behr, Jean-Paul; Filhol, Odile; Pares, Serge; Garin, Jerome; Bourgarit, Jean-Jacques; Chambaz, Edmond M.; Cochet, Claude [Reprint author]
CS Commissariat Energie Atomique, Biochmie des Regulations Cellulaires Endocrines, INSERM Unit 244, F-38054 Grenoble Cedex 9, France
SO Journal of Biological Chemistry, (**1995**) Vol. 270, No. 29, pp. 17400-17406.
AB Phosphorylation of many protein substrates by the protein **kinase** casein **kinase** 2 (CK2) is stimulated severalfold in the presence of polyamines such as spermine. Previous experiments have shown that CK2 is a

polyamine binding protein and that the regulatory beta subunit is required for this binding activity. To delineate the spermine binding site of CK2, we have applied a photoaffinity labeling method using a tritiated photoactivable analog of spermine, (3H)sperminediazonium. The photoaffinity labeled beta subunit was cleaved with cyanogen bromide, and **two labeled peptides** were separated by high performance liquid chromatography. The major one was the peptide T-72EQAAEM-78 and the minor one was a 22-amino acid peptide comprising residues Ile-98 to Met-119. Thr-72 and His-06 were identified as the labeled amino acids of the Thr-72-Met-78 and Ile-98Met-119 peptides, respectively. In the same manner, we succeeded in determining the residue Leu-220 as an a subunit residue covalently bound to the probe. The photoaffinity labeling method described here enabled the first elucidation, by direct microsequencing, of a polyamine binding site on CK2 for which we propose a provisional structural model. These observations suggest a possible mechanism for CK2 activation by polyamines at the molecular level.

L8 ANSWER 6 OF 31 BIOSIS on STN
AN 1995:166361 BIOSIS
TI Membrane domains containing phosphatidylserine and substrate can be important for the activation of protein **kinase** C.
AU Yang, Li; Glaser, Michael [Reprint author]
CS Dep. Biochemistry, Univ. Ill., 600 South Mathews Avenue, Urbana, IL 61801, USA
SO Biochemistry, (**1995**) Vol. 34, No. 5, pp. 1500-1506.
AB The relationship between lipid domains and enzyme activity was studied via the direct visualization and quantitation of domains by fluorescence digital imaging microscopy. The substrate used in these experiments was a basic peptide derived from a prominent cellular substrate (MARCKS) of protein **kinase** C. The MARCKS **peptide** and phosphatidylserine, which were **labeled** by **two** different fluorophores, colocalized into domains in large vesicles (5-10 mu-m). Increasing the ionic strength disrupted the domains of the MARCKS peptide and phosphatidylserine, and this was accompanied by a decrease in protein **kinase** C activity. Dansylpolylysine, which inhibits protein **kinase** C, was similar to the MARCKS peptide in forming domains enriched in phosphatidylserine. The degree of enrichment of the MARCKS peptide in the phosphatidylserine domains decreased proportionally with protein **kinase** C activity when polylysine was added. Polylysine caused the MARCKS peptide to be displaced from the domains into the nondomain areas of the vesicles. This suggested that binding of the substrate to the vesicles was not the critical factor for protein **kinase** C activity, but rather it was the organization of the substrate into domains that was related to the activation of the enzyme. Gramicidin, which was chosen to represent a neutral membrane protein, was excluded from the domains with phosphatidylserine, and it had no effect on the enrichment of the domains or the enzyme activity. The results of this study show that the formation of membrane domains can be important for the activation of protein **kinase** C and the activity can be inhibited by disrupting the domains.

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STN INTERNATIONAL LOGOFF AT 14:03:24 ON 20 DEC 2006